A Deterministic Mathematical Model for Ebola Virus incorporating the Vector Population

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***Abstract* —** *Most Mathematical model for Ebola virus in the literature had only the human population. This paper is an attempt to incorporate the host population which will give a clearer view of the transmission dynamics of the deadly disease. The disease free and endemic equilibrium of the model were obtained and analyzed for stability, . Key to our analysis is the basic reproductive number* R_0 *which is the number of secondary infections that one infective individual would create over the duration of the infectious period provided that everyone else is susceptible. We computed a numerical value for* R_0 *and conducted a sensitivity analysis of its parameters. Our results reveal that quarantine of infected individual's speeds up recovery time.*

Keywords — *Key words: Stability, Equilibrium, Quarantine, Host, and Endemic*

1. INTRODUCTION

A complex epidemic of *Zaire ebolavirus* (EBOV) has been affecting West Africa since December 2013, with the first cases likely occurring in southern Guinea [1]. Ebola first appeared in 1976 in 2 simultaneous outbreaks, in Nzara, Sudan, and in Yambuku, Democratic Republic of Congo. The latter was in a village situated near the Ebola River, from which the disease takes its name

The first victim, and the index case for the disease, was village school headmaster Mabalo Lokela, who had toured an area near the Central African Republic border along the Ebola river between 12–22 August. On 8th September he died of what would become known as the Ebola virus [2]. Subsequently a number of other cases were reported, almost all centered on the Yambuku mission hospital or having close contact with another case. 318 cases and 280 deaths (a 88% fatality rate) occurred in the DRC [3]. The disease is endemic in some wes African countries including Uganda, Sierra Loene, Liberia, Giunea Nigeria etc. [4]

Ebola is introduced into the human population through close contact with the blood, secretions, organs or other bodily fluids of infected animals. In Africa, infection has been documented through the handling of infected chimpanzees, gorillas, fruit bats, monkeys, forest antelope and porcupines found ill or dead or in the rainforest.

Ebola then spreads in the community through human-to-human transmission, with infection resulting from direct contact (through broken skin or mucous membranes) with the blood, secretions, organs or other bodily fluids of infected people, and indirect contact with environments contaminated with such fluids [5]. Burial ceremonies in which mourners have direct contact with the body of the deceased person can also play a role in the transmission of Ebola [6].

EBOV is a severe acute viral illness often characterized by the sudden onset of fever, intense weakness, muscle pain, headache and sore throat. This is followed by vomiting, diarrhoea, rash, impaired kidney and liver function, and in some cases, both internal and external bleeding. The incubation period, that is, the time interval from infection with the virus to onset of symptoms, is 2 to 21 days.

Ebola virus infections can be diagnosed definitively in a laboratory through several types of tests: antibody-capture enzyme-linked immunosorbent assay (ELISA), antigen detection tests, serum neutralization test, reverse transcriptase polymerase chain reaction (RT-PCR) assa, electron microscopy, virus isolation by cell culture, etc

No licensed vaccine for EVD is available. Several vaccines are being tested, but none are available for clinical use, and no specific treatment is available. New drug therapies are being evaluated.

Mathematical model has been an important tool in analyzing the spread and control of infectious diseases. The first of such models was credited to Daniel Bernoulli in 1760, the aim of his model was to evaluate the impact of variolation or inoculation on healthy people with smallpox virus [7]. This was followed by [8] model on measles, [9] model on malaria, and the famous[10] SIR model for the transmission of disease in a closed population. The past century has witnessed rapid development of mathematical models to understand the dynamics of infectious diseases.

Refenence [11] constructed a model describing the spread of the deadly disease called Ebola hemorrhagic fever. The model was first constructed using the classical derivative and then converted to the generalized version using the beta derivative. He

studied in detail the endemic equilibrium points and provided the Eigen values associated using the Jacobian method.

The study showed that, for small portion of infected individuals, the whole country could die out in a very short period of time in case there is no good prevention.

Reference [12] represented the transmission of Ebola virus using a modified Susceptible – Infected – Recovered (SIR) disease model. The model had four compartments, $S(t)$, $I(t)$, $R(t)$, and $D(t)$ representing the Susceptible, Infective, Recovered and the Deceased compartment. The parameters of the model are a the rate of infection, b the rate of recovery, c the rate of Susceptibility, e the rate of death.

Similarly other recent models [13],[14],[15] centered on the estimation of basic reproduction number, a key threshold in disease control but lack other further mathematical analysis

II. MATERIAL AND METHOD

In this section the details of the model formulation as well as the assumptions will be given.

A. Model Formulation

The Infected population is generated via birth and immigration, is decreased by infection with Infected human or vector and natural death. The Exposed population is generated when there is effective contact between the Suceptible Human and any of Infected Human, Quarantine or the infected Vector. It is decreased by either naturally death since they do not manifest symptoms or progresses to the infected population. The Infected population is either isolated to the quarantine center, die naturally or as nresult of the virus. The Quarantine recovers and move to the Recovered group, die naturally or due to the virus. The Recovered population acquire lifelong immunity and can only die naturally. The Susceptible vector is generated by natural birth and is decreased via contact with Infected vector and natural death, we ignored exposed population in the vector population. Using these assumptions, we present our model as follows

$$\frac{dS}{dt} = \Lambda_H - \alpha_1(I + \eta Q + I_R)S - \mu_1 S \quad (1)$$

$$\frac{dE}{dt} = \alpha_1(I_H + \eta Q + I_R)S - (\mu_1 + \sigma_1)E \quad (2)$$

$$\frac{dI_H}{dt} = \sigma_1 E - (\sigma_2 + \mu_1 + \delta_1)I_H \quad (3)$$

$$\frac{dQ}{dt} = \sigma_2 I_H - (\mu_1 + \delta_1 + \gamma)Q \quad (4)$$

$$\frac{dR}{dt} = \gamma_1 Q - \mu_1 R \quad (5)$$

$$\frac{dS_R}{dt} = \Lambda_R - \alpha_2 I_H S_R - \mu_2 S_R \quad (6)$$

$$\frac{dI_R}{dt} = \alpha_2 I_R S_R - (\mu_2 + \delta_2)I_R \quad (7)$$

Adding equations (1) to (5) gives

$$\frac{dN_H}{dt} = \Lambda_H - \mu_1 N_H - \delta(I + Q) \quad (8)$$

Also, adding equations (6) and (7) gives

$$\frac{dN_R}{dt} = \Lambda_R - \mu_2 N_R - \delta I_R \quad (9)$$

B. Symbols and Paramters

The symbols and paramters used in the model are listed below:

S_H	Susceptible human population
E	Exposed human population
I_H	Infected human pouplation
Q	Quarantine human population
R	Recovered Human population
S_V	Susceptible Vector population
I_V	Infected vector population
Λ_H	Recruitment rate of human population
Λ_V	Birth rate of vector population
α_1	Effective contact rate of human
η	Modification parameter
α_2	Effective contact rate for vector
σ_1	Progression rate from exposed to infected
σ_2	Transffere rate from infected to quarantine
γ	Recovery rate of the quarantine class
μ_1	Per capital natural death rate of human
μ_2	Per capital natural death rate of vector
δ_1	Death rate of human due to Ebola virus
δ_2	Death rate of the vector due to Ebola virus

C. Positivity of Solution

In the absence of the disease, the total human population and the total vector population sizes ,

approaches respectively the carrying capacities $\frac{\Lambda_H}{\mu_1}$, and $\frac{\Lambda_R}{\mu_2}$. The differential equations for N_H and N_R given by equations (8) and (9) implies that the solution of equations (1) to (7) starting in the positive orthan R_8^+ approaches, enter, or remains in the epidemiologically meaningful subset D.

Where

$$D = \left\{ \begin{array}{l} (S,E,I,Q,R,S_R,I_R)/(S>0, V \geq 0, E \geq 0, I \geq 0, Q \geq 0, R \geq 0, S_R > 0, I_R \geq 0). S+E+I \\ +Q+R \leq \frac{\Lambda_H}{\mu_1}, S_R + I_R \leq \frac{\Lambda_R}{\mu_2} \end{array} \right\}$$

Thus it suffices to consider solution in region D. Solution of the initial value problem starting in D and defined by equations (1) to (7) exist and is unique on maximal interval. Since solution remains bounded in the positively invariant region D, the maximal interval is $(0,\infty)$. Thus, the initial value problem is well posed both mathematically and epidemiologically.

III. RESULTS

A. *Disease Free Equilibrium*

The disease free equilibrium of our model equations (1) to (7) is given by

$$E_0\left(S_H^*, E, I_H^*, R, Q, S_R^*, I_R^*\right) = \left(\frac{\Lambda_H}{\mu_1}, 0, 0, 0, 0, \frac{\Lambda_R}{\mu_2}, 0\right) \quad (10)$$

The stability of this disease free equilibrium given by equation (10) will be analyzed via the basic reproductive number

B. *The Basic Reproductive Number* (R_0)

One of the most important concerns about any infectious disease is its ability to invade a population. Many epidemiological models have a disease free equilibrium (DFE) at which the population remains in the absence of the disease. These models usually have a threshold parameter, known as the basic reproductive number R_0 such that when $R_0 \leq 1$, then the DFE is locally asymptotically stable, and the disease cannot invade the population, but if $R_0 > 1$, then the DFE is unstable and invasion is always possible see [16].

We define the basic reproductive number R_0 as the number of secondary infections that one infective individual would create over the duration of the infectious period provided that everyone else is susceptible. Our model is suited for a heterogeneous population in which the vital and epidemiological parameter for an individual may depend on such factors as the stage of the disease, spatial position, etc. however, we assume that the population can be broken into homogeneous subpopulation or compartment such that individual in a given compartment are indistinguishable from one another.

The next generation matrix approach as described by [17] was used to derive our Basic Reproductive Number R_0. Numerous other articles [18] – [20], are devoted to the calculation of basic reproductive number R_0 for different models of various diseases.

Here, the basic reproductive number R_0 is the spectral radius (dominant eigenvalue) of the product matrix FV^{-I}, i.e $R_0 = \rho(FV^{-I})$

Our model has three Infective compartments namely the Exposed Human E Infectious I_H, Quarantine Human Q and Infected Reservoir I_H compartments. It follows that the matrices F and V are for the new infective terms and remaining transfer terms respectively are given below. Where the entries of F and V are partial derivatives of $f_i(x)$ and $v_i(x)$. For our model, F and V are given below.

$$F = \begin{bmatrix} 0 & \alpha_1 S_H^* & \eta\alpha_1 S_H^* & \alpha_1 S_H^* \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha_2 S_R^* \end{bmatrix}$$

$$V = \begin{bmatrix} K_1 & 0 & 0 & 0 \\ -\sigma_1 & K_2 & 0 & 0 \\ 0 & -\sigma_2 & K_3 & 0 \\ 0 & 0 & 0 & K_4 \end{bmatrix}$$

$$V^{-1} = \begin{bmatrix} \frac{1}{K_1} & 0 & 0 & 0 \\ -\frac{\sigma_1}{K_1 K_2} & \frac{1}{K_2} & 0 & 0 \\ \frac{\sigma_1\sigma_2}{K_1K_2K_3} & -\frac{\sigma_2}{K_2K_3} & \frac{1}{K_3} & 0 \\ 0 & 0 & 0 & \frac{1}{K_4} \end{bmatrix}$$

$$FV^{1} = \begin{bmatrix} \frac{\sigma_1\alpha_1 S_H^*(\sigma_2\eta - K_3)}{K_1K_2K_3} & \frac{\alpha_1 S_H^*(K_3\sigma_2\eta)}{K_2K_3} & \frac{\alpha_1\eta S_H^*}{K_3} & \frac{\alpha_1 S_H^*}{K_4} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\alpha_2 S_R^*}{K_4} \end{bmatrix}$$

where

$K_1 = \mu_1 + \sigma_1,\ K_2 = \sigma_2 + \mu_1 + \delta_1,$

$K_3 = \gamma + \mu_1 + \delta_1,\ K_4 = \mu_2 + \delta_2 - \alpha_2 S_R^*$

Tthe basic reproductive number R_c is the spectral radius (dominant eigenvalue) of the product matrix FV^{-I}, that is

$$R_0 = \rho(FV^{-I}) = \frac{\sigma_1\alpha_1\Lambda_H\{\eta\sigma_2 - (\gamma + \mu_1 + \delta_1)\}}{\mu_1(\mu_1 + \sigma_1)(\sigma_2 + \mu_1 + \delta_1)(\gamma + \mu_1 + \delta_1)} \quad (11)$$

C. *Stability of the Disease Free Equilibrium State*

At disease free equilibrium the Jacobian of the equations (1) to (7) is

$$J_{E_0} = \begin{bmatrix} -\mu_1 & 0 & S_H^*\alpha_1 & S_H^*\eta\alpha & 0 & 0 & S_H^*\tau\alpha \\ 0 & -(\mu_1+\sigma_1) & 0 & 0 & 0 & 0 & 0 \\ 0 & \sigma_1 & -(\mu_1+\sigma_2+\delta_1) & 0 & 0 & 0 & 0 \\ 0 & 0 & \sigma_2 & -(\mu_1+\delta_1+\gamma) & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma & -\mu_1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu_2 & -\alpha_2 S_R^* \\ 0 & 0 & 0 & 0 & 0 & 0 & -(\mu_2+\delta_2-\alpha_2 S_R^*) \end{bmatrix}$$

(12)

The eigen values of (12) are

$-\mu_1, -(\mu_1+\sigma_1), -(\sigma_2+\mu_1+\delta_1), -(\gamma_1+\mu_1+\delta_1), -\mu_2,$

$-\left(\mu_2+\delta_2-\alpha_2 S_R^*\right)$

Which are all n egative, hencethe the system of model equations (1) to (7) is locally asymptotically stable at disease free equilibrium point E_0.

Lemma 1.

If $R_0 < 1$, the disease free equilibrium point E_0 is locally asymptotically stable. If $R_0 = 1$, E_0 is stable. If $R_0 > 1$, E_0 is unstable.

Let,

$$f_\infty = \lim_{t\to\infty}\inf_{\theta\geq t} f(\theta)$$

$$f^\infty = \lim_{t\to\infty}\sup_{\theta\geq t} f(\theta)$$

Lemma 2.

Assume that a bounded real valued function $f:[0,\infty]\to R$ be twice differentiable with bounded second derivative. Let $k\to\infty$ and $f(t_k)$ converges to f^∞ to f_∞ then, $\lim_{t\to\infty} f'(t_k) = 0$

Theorem 1.

if $R_0 < 1$ then worm free equilibrium E_0 is globally asymptotically stable.

Proof.

From the system of model equation (1) to (7) we have,

$$\frac{dS}{dt} \leq \Lambda_H - \mu_1 S$$

A solution of the equation $\frac{dX}{dt} \leq \Lambda_H - \mu_1 X$ is a super solution of $S(t)$.

Since $X(t) \to \frac{\Lambda_H}{\mu_1}$ as $t\to\infty$, then for given $\in > 0$, such that $S(t) \leq X(t) \leq \frac{1}{\mu_1} + \in$ for $t \geq t_0$

Thus, $S^\infty \leq \frac{\Lambda_H}{\mu_1} + \in$.

Let $\in \to 0$ then, $S^\infty \leq \frac{\Lambda_H}{\mu_1}$

Similarly, equation (2) can be expressed as

$$\frac{dE}{dt} = \alpha_1(I_H + \eta Q + I_R)\frac{\Lambda_H}{\mu_1} - (\mu_1 + \sigma_1)E \quad (13)$$

Using (13), and equations (3), (4), and (7) of our model, we have

$$\begin{bmatrix} \dot{E} \\ \dot{I}_H \\ \dot{Q} \\ \dot{I}_R \end{bmatrix} \leq A \begin{bmatrix} E \\ I_H \\ Q \\ I_R \end{bmatrix}$$

Where

$$A=\begin{bmatrix} -(\mu_1+\sigma_1) & 0 & 0 & 0 \\ \sigma_1 & -(\mu_1+\sigma_2+\delta_1) & 0 & 0 \\ 0 & \sigma_2 & (\mu_1+\delta_1+\gamma) & 0 \\ 0 & 0 & 0 & -\left(\mu_2+\delta_2-\alpha_2\frac{\Lambda_R}{\mu_2}\right) \end{bmatrix}$$

(14)

Let $M \in R^+$, such that

$$M>\max\left\{(\mu_1+\sigma_1),(\sigma_2+\mu_1+\delta_1),(\gamma_1+\mu_1+\delta_1),\mu_2,\left(\mu_2+\delta_2-\alpha_2\frac{\Lambda_R}{\mu_2}\right)\right\}.$$

Thus $A+MI_{4\times4}$ is a strickly positive matrix where I_{4X4} is an identity matrix.

If w_1, w_2, w_3 and w_4 are the eigen values of A then $w_1+M, w_2+M, w_3+M, w_4+M$ are the eigen values of $A+MI_{4\times4}$.

Thus from the perron-Frobenius theorem [ ] $A+MI_4$ has a simple positive eigen value equal to dominant eigen value and corresponding eigen vector $e>0$ which implies that w_1, w_2, w_3 and w_4 are real. If w_1+M is the dominant eigen value of $A+MI_{4\times4}$, then $w_1>w_2, w_1>w_3, w_1>w_4$ and $eA>ew_1$. Obviously, w_1, w_2, w_3 and w_4 are the roots of the equation:

$$w^4+[a-(g+f+c)]w^3+[a(g+f+c)+c(g+f)-gf]w^2+[a(c(g+f)+gf)+cgf]w-cgfa=0$$

(15)

where

$a=(\mu_1+\sigma_1)$, $c=(\mu_1+\sigma_2+\delta_1)$,

$g=(\mu_1+\delta_1+\gamma)$, $f=\left(\mu_2+\delta_2-\alpha_2\frac{\Lambda_R}{\mu_2}\right)$

Since $R_0<1$ for $\in>0$, sufficiently small, we have,

$$(\mu_1+\sigma_1)\,(\mu_1+\sigma_2+\delta_1)\,(\mu_1+\delta_1+\gamma)\left(\mu_2+\delta_2-\alpha_2\frac{\Lambda_R}{\mu_2}\right)>0$$

Therefore, coefficients of the quadratic equation (15) are positive. Thus, w_1, w_2, w_3, and w_4 are negative. So from equation (14) for t ⍰ t_0

$$\frac{d}{dt}\left(e.[E(t),I_H(t),Q(t),I_R(t)]\right)\leq w_1.e[E(t),I_H(t),Q(t),I_R(t)]$$

Integrating the above inequality, we get,

$$0\leq e.[E(t),I_H(t),Q(t),I_R(t)]\leq e.[E(t),I_H(t),Q(t),I_R(t)]e^{w_1(t-t_1)}$$

for $t\geq t_1\geq t_0$:

Since $w_1<o, e.[E(t),I_H(t),Q(t),I_R(t)]\to 0$ as $t\to\infty$ $\omega_1<0$, $e\cdot[E(t_1), I(t_1), Q(t_1)]\to 0$ as $t\to\infty$

Using $e>0$, we have,

$$[E(t),I_H(t),Q(t),I_R(t)]\to(0000) \text{ as } t\to\infty$$

By Lemma 2 we choose a sequence

$t_n\to\infty, S_n\to\infty(n\to\infty)$ such that

$$S(S_n)\to S^\infty, S(t_n)\to S_\infty, S(S_n)\to 0, S(t_n)\to 0$$

Since $E(t), I_H(t)\to Q(t)\to 0$ as $t\to\infty$) thus from the first equation of our model (1), we have,

$$\lim_{n\to\infty} S(t)=\frac{\Lambda_H}{\mu_1}$$

Hence, by incorporating Lemma 1, the disease free equilibrium E_0 is globally asymptotically stable, if $R_0<1$.

D. *Existence Endemic Equilibrium State*

In order to find the endemic equilibrium of the our model equations given by equations (1) to (7) i.e. equilibria where at least one of the infected components of the model is non zero, the following steps are taken. We let $E_1=\left(S_H^{**}, E^{**}, I_H^{**}, Q^{**}, R^{**}, S_R^{**}, I_R^{**}\right)$ represent any arbitrary endemic equilibrium of our model equations (1) to (7). Further, let

$$\pi=\alpha_1(I_H+\eta Q+I_V) \quad (16)$$

Be the force of infection for human, at steady state. Substituting (16) into equations (1) to (5) and solving at steady state, we have

$$S_H^{**} = \frac{\Lambda_H}{\pi + \mu_1} \quad (17)$$

$$E^{**} = \frac{\pi\Lambda_H}{(\pi+\mu_1)(\pi+\sigma_1)} \quad (18)$$

$$I_H^{**} = \frac{\pi\Lambda_H\sigma_1}{(\pi+\mu_1)(\pi+\sigma_1)(\sigma_2+\mu_1+\delta_1)} \quad (19)$$

$$Q^{**} = \frac{\pi\Lambda_H\sigma_1\sigma_2}{(\pi+\mu_1)(\pi+\sigma_1)(\sigma_2+\mu_1+\delta_1)(\mu_1+\delta_1+\gamma)} \quad (20)$$

$$R^{**} = \frac{\pi\Lambda_H\sigma_1\sigma_2\gamma}{(\pi+\mu_1)(\pi+\sigma_1)(\sigma_2+\mu_1+\delta_1)(\mu_1+\delta_1+\gamma)\mu_1} \quad (21)$$

from equations (6) and (7), we have

$$S_V^{**} = \frac{(\mu_2+\delta_2)\Lambda_V}{\mu_2} \quad (22)$$

$$I_V^{**} = \frac{\mu_2}{\alpha_2 - \Lambda_V} \quad (23)$$

Substituting (19), (20) and (23) into (16), and simplifying, we have the quadratic equation

$$\pi^2 + \pi(\alpha_2 - \Lambda_V)\left[\begin{array}{l}\{(\mu_1(\mu_1+\sigma_1)(\sigma_2+\mu_1+\delta_1)(\mu_1+\delta_1+\gamma))\\ (1+(\mu_2+\delta_2)\mu_2)\\ -\alpha_1\Lambda_H\sigma_1((\mu_1+\delta_1+\gamma)+\sigma_2\end{array}\right] = 0 \quad (24)$$

Solving (24) for π and substituting into (17) to (21) gives explicit values for the various human compartment

$S_H^{**}, E^{**}, I_H^{**}, Q^{**}, R^{**}$ as S_R^{**}, I_R^{**} are already explicit

Table 1. Parameter values and initial conditions

S/No	Parameters	Values
1	Λ_H	**0.3**
2	α_1	**0.016**
3	η	**0.8**
4	μ_1	**0.2**
5	σ_1	**8**
6	δ_1	**0.6**
7	γ	**0.3**
8	σ_2	**6**
9	Λ_R	**0.65**
10	μ_2	**0.6**
11	δ_2	**0.5**
12	α_2	**0.02**

E. Numerical Simulation

Figure 1is a numerical simulation of the Ebola virus model given by equations (1) to (7), using the original system variables with parameter values as given in table 1. The simulations were conducted using the Runge-Kuta method (rkf45) embedded in Maple 13. The rkf45 method is a fourth-order method, meaning that the local truncation error is on the order of $0(h^5)$, while the total accumulated error is order $0(h^4)$.

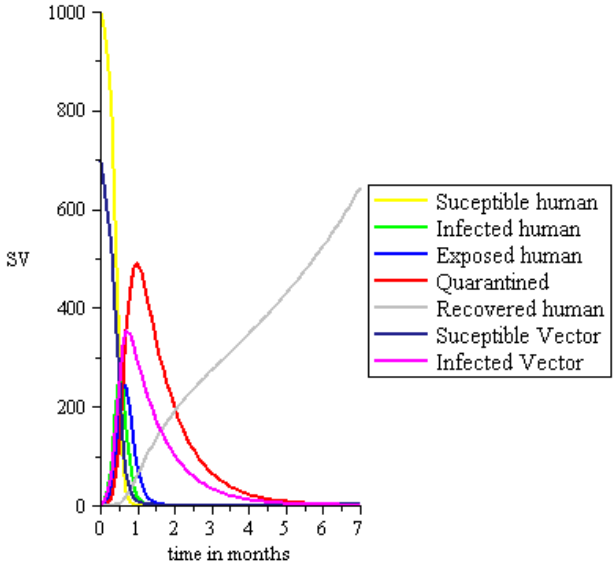

Fig. 1 Dynamics of the state variables (sv) with respect to time, paramter values as shown in table 1

F. Computation of R_0 Sensitivity Analysis

Our effective reproductive number R_0 is given by equation (11), the numerical value is computed to be $R_0 = 0.011582105$1, the maple 13 code used is presented in appendix A. The parameters of R_0 are $\Lambda_H, \eta, \alpha_1, \mu_1, \delta_1, \sigma_1, \gamma$, and σ_2, their sensitivity tells us how important they to disease transmission. Such information, is crucial not only to experimental design, but also to data assimilation and reduction of complex nonlinear model [21]. Sensitivity Analysis is commonly used to determine the robustness of model prediction to parameter values, since there are usually errors in data collection and presumed parameter values. It is used to determine parameters that have high impact on the R_C and should be targeted by intervention strategies.

G. Computation of R_0 Sensitivity Analysis

Our effective reproductive number R_0 is given by equation (11), the numerical value is computed to be

$R_C = 0.0115821051$, the maple 13 code used is presented in appendix A. The parameters of R_0 are $\Lambda_H, \eta, \alpha_1, \mu_1, \delta_1, \sigma_1, \gamma,$ and σ_2, their sensitivity tells us how important they to disease transmission. Such information, is crucial not only to experimental design, but also to data assimilation and reduction of complex nonlinear model [21].

Sensitivity Analysis is commonly used to determine the robustness of model prediction to parameter values, since there are usually errors in data collection and presumed parameter values. It is used to determine parameters that have high impact on the R_0 and should be targeted by intervention strategies.

Given the explicit formula for R_0 one can easily derive an analytical expression for the sensitivity of R_0 with respect to each parameter that comprises it. Table 2 below presents the sensitivity index for the paramters of our R_0, the index table reveals that the most sensitive parameter to our Effective Reproductive number is η. The maple code that generated the sensitivity index is too lentghy to be appended

Sensitivity Analysis Index for R_0 Paramters

S/no	Parameters	Sign	Values
1	Λ_H	+	**1**
2	α_1	+	**1**
3	η	+	**1.297**
4	μ_1	-	**1.29**
5	σ_1	+	**0.024**
6	δ_1	-	**0.796**
7	γ	-	**0.354**
8	σ_2	+	**0.415**

H. Conclusion

In this paper a deterministic mathematical model for the dynamics of Ebola Virus was formulated. The model incorporated a vector population, and the rate of transmission from the quarantined to the susceptible was weight as a result of restriction of movement imposed on the quarantined territory. We first showed that our model is epidemiologically and mathematically well posed. Further, we obtained both the disease and endemic equilibria and analyzed them for stability. It was established that the disease free equilibrium is both locally and globally stable. We obtained the numerical value for R_0 and conducted the sensitivty analysis of its variables. finally we depicted a graph of the various state variable against time

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Appendix A

Computation of Basic Reproductive Number R_C

```
> restart
> with(Basic Reproductive number, R0)
    with(Basic Reproductive number, R0)
>
Λ[H] := 0.3; α[1] := 0.016; η := 0.8; μ[1] := 0.2; σ[1] := 8; δ[1] := 0.6; τ := 0.3; σ[2] := 6
```

$$\Lambda_H := 0.3$$

$$\alpha_1 := 0.016$$

$$\eta := 0.8$$

$$\mu_1 := 0.2$$

$$\sigma_1 := 8$$

$$\delta_1 := 0.6$$

$$\tau := 0.3$$

$$\sigma_2 := 6$$

```
>
R0 := (σ[1]·α[1]·Λ[H]·(η·σ[2] − (τ + μ[1] + δ[1])))/(μ[1]·(μ[1] + σ[1])·(σ[2] + μ[1] + δ[1])·(τ + μ[1] + δ[1]))
```

$$R_0 := 0.0115821051$$